

**U.S. Department of Labor**

Office of Administrative Law Judges  
525 Vine Street, Suite 900  
Cincinnati, OH 45202

Telephone: (513) 684-3252  
Facsimile: (513) 684-6108



Date Issued: August 22, 2000

Case No: 1999-BLA-1133

In the Matter of

ALVIN H. STANLEY  
Claimant

v.

HUMPHREYS ENTERPRISES, INC.  
Employer

CNA INSURANCE COMPANY  
Carrier

and

DIRECTOR, OFFICE OF WORKERS' COMPENSATION PROGRAMS  
Party-in-Interest

**APPEARANCES:**

Lawrence L. Moise, III, Esq.  
For the claimant

Timothy W. Gresham, Esq.  
For the employer and carrier

BEFORE: JOSEPH E. KANE  
Administrative Law Judge

**DECISION AND ORDER - AWARDING BENEFITS**

This proceeding arises from a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, 30 U.S.C. § 901 *et seq.* (the Act). Benefits are awarded to coal miners who are totally disabled due to pneumoconiosis. Surviving dependents of coal miners whose deaths were caused by pneumoconiosis may also recover benefits. Pneumoconiosis, commonly known as black lung, is a chronic dust disease of the lungs arising from coal mine employment. 20 C.F.R. § 718.201 (1996).

On July 20, 1999, this case was referred to the Office of Administrative Law Judges for a formal hearing. A hearing on December 16, 1999 before Administrative Law Judge Daniel Roketenetz was continued at the Claimant's request.

Following proper notice to all parties, a hearing was held before the undersigned on April 11, 2000 in Abingdon, Virginia. The Director's exhibits were admitted into evidence pursuant to 20 C.F.R. § 725.456, and the parties had full opportunity to submit additional evidence and to present closing arguments or post-hearing briefs. The Employer's Closing Argument was received on June 1, 2000. The Claimant's Closing Comments was received and accepted on July 6, 2000.

The Findings of Fact and Conclusions of Law that follow are based upon my analysis of the entire record, arguments of the parties, and the applicable regulations, statutes, and case law. They also are based upon my observation of the demeanor of the witness who testified at the hearing. Although perhaps not specifically mentioned in this decision, each exhibit and argument of the parties has been carefully reviewed and thoughtfully considered. While the contents of certain medical evidence may appear inconsistent with the conclusions reached herein, the appraisal of such evidence has been conducted in conformance with the quality standards of the regulations.

The Act's implementing regulations are located in Title 20 of the Code of Federal Regulations, and section numbers cited in this decision exclusively pertain to that title. References to DX, CX and EX refer to the exhibits of the Director, the claimant, and the employer, respectively. The transcript of the hearing is cited as "Tr." and by page number.

### ISSUES

The following issues remain for resolution:

1. whether the claimant has pneumoconiosis as defined by the Act and regulations;
2. whether the claimant has complicated pneumoconiosis;
3. whether the claimant is totally disabled; and
4. whether the claimant's disability is due to pneumoconiosis.

(Tr. 11-13; DX 28). The employer does not contest that any simple pneumoconiosis the claimant has arose from his coal mine employment. (Employer's Closing Argument at p. 7, n. 5).

## FINDINGS OF FACT AND CONCLUSIONS OF LAW

### Factual Background and Procedural History

The claimant, Alvin Harold Stanley, was fifty-one years old at the time of the hearing and has a fifth or sixth grade education. He has three dependents, his wife and two minor children, for purposes of augmentation of benefits. (Tr. 14-15, 28; DX 1, 7, 8, 9). The claimant is currently receiving Social Security disability benefits. He is blind in his right eye and suffered a back injury, both due to accidents on the job. (Tr. 26; EX 23, 24, 25).

The parties stipulated that the claimant had at least twenty years of coal mine employment. (Tr. 12, 13). I accept that stipulation as supported by the evidence of record. (DX 2, 5). The claimant last worked in April of 1998, as a drill operator and hauler operator on a strip mine, for the named employer. (Tr. 15, 24-25, 37; DX 3, 4). All of his coal mine employment was in the state of Virginia. (Tr. 26; DX 5). The claimant testified that he tried using respirators while he worked, but that due to a skin reaction, he could not wear them. At times, he used a cloth on his face. (Tr. 25-26).

The claimant also testified that he began experimenting with cigarettes at the age of ten. He did not begin smoking on a regular basis until approximately age fifteen or sixteen. He usually smoked one pack a day. At one point, however, he did use up to four packs of cigarettes per day, although the claimant stated that about half of those cigarettes were smoked by his boss who "bummed" them off of him. (Tr. 26-27; EX 23, 25).

The claimant filed his claim for benefits under the Act on November 17, 1998. (DX 1). The employer was notified of the claim, and subsequently controverted based on both its liability and the claimant's eligibility. (DX 16, 17, 18, 19, 20). The District Director, Office of Workers' Compensation Programs ("OWCP") awarded benefits on June 11, 1999. (DX 25). As the employer declined to voluntarily commence the payment of benefits, benefits have been paid by the Black Lung Disability Trust Fund retroactive to June 1, 1999. (DX 27). The employer timely requested a formal hearing, and the case was referred to the Office of Administrative Law Judges ("OALJ") on July 20, 1999. (DX 26, 29).

Medical Evidence

A. Chest X-rays

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u> <sup>1</sup>	<u>Interpretation</u>
DX 23	1/11/83	-	Maddox (Hosp.)	Heart is not enlarged. Hila lungs and mediastinum are within normal limits. No interval change when compared to 12-14-82.
CX 3	7/12/95	-	DePonte/BCR, B (Hospital)	Changes of advanced pneumoconiosis with multiple rounded and irregular opacities in the lung apices with coalescence of the opacities and apical volume loss. No evidence of pneumonia. Calcifications in the hilar lymph nodes, more so on the left.

---

<sup>1</sup>The symbol "BCR" denotes a physician who has been certified in radiology or diagnostic roentgenology by the American Board of Radiology, Inc. or the American Osteopathic Association. 20 C.F.R. § 727.206(b)(2).

The symbol "B" denotes a physician who was an approved "B-reader" at the time of the x-ray reading. A B-reader is a physician who has demonstrated expertise in assessing and classifying x-ray evidence of pneumoconiosis. These physicians have been approved as proficient readers by the National Institute of Occupational Safety & Health, U.S. Public Health Service pursuant to 42 C.F.R. § 37.51 (1982).

<b><u>Ex.No.</u></b>	<b><u>Date of X-ray</u></b>	<b><u>Film Qual.</u></b>	<b><u>Physician/ Qualifications</u></b>	<b><u>Interpretation</u></b>
EX 11	7/12/95	2	Wheeler/BCR, B	Ill defined mass or infiltrate fibrosis in apices and posterolateral portion RUL > LUL compatible with conglomerate Tb. Subtle interstitial infiltrate in upper lobes due to Tb unknown activity, probably healed with small scattered minimal fibrosis or adenopathy left hilum with probable calcified granulomata. Hi. Tb.
CX 3	4/20/98	-	DePonte/BCR, B (Hospital)	Pneumoconiosis with progressive massive fibrosis. Compared with x-ray of 7/12/95.
EX 13	4/20/98	2	Wheeler/BCR, B	1.5 x 2 cm. calcified granulomata left hilum seen better on lateral view since last exam 7/12/95 compatible with healed Tb of histoplasmosis. Hi.
CX 3	4/22/98	-	DePonte/BCR, B (Hospital)	Pneumoconiosis with progressive massive fibrosis, unchanged.
EX 14	4/22/98	2	Wheeler/BCR, B	Unchanged except for technique since last exam 4/20/98.
CX 3	4/30/98	-	DePonte/BCR, B	Changes of pneumoconiosis

(Hospital)

with progressive massive fibrosis, unchanged from 4/22/98.

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/Qualifications</u>	<u>Interpretation</u>
EX 12	4/30/98	1	Wheeler/BCR, B	Ill defined infiltrate or fibrosis mass in apices and lateral RUL > LUL compatible with conglomerate Tb with calcified granulomata in both lungs and left hilar adenopathy. The disease extends to pleura and is mainly in posterior portion upper lobes.
CX 3	5/7/98	-	DePonte/BCR, B (Hospital)	Pneumoconiosis with progressive massive fibrosis, unchanged from 4/30/98.
EX 15	5/7/98	1	Wheeler/BCR, B	Increased density in mass or fibrosis right apex compatible with calcified granulomata in conglomerate Tb. Hi.
EX 16	5/7/98	1	Scott/BCR, B	0/1, p/s, mid and upper. Calcified granulomata. Peripheral upper lung infiltrates. Probable peripheral granulomatous mass right apex. Change compatible with healed Tb $\pm$ activity. Em; hi.

CX 3	5/9/98	-	DePonte/BCR, B (Hospital)	Pneumoconiosis with progressive massive fibrosis, unchanged from 5/7/98.
EX 1	5/9/98	2	Wheeler/BCR, B	Unchanged except for technique since last exam 5/7/98.

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u>	<u>Interpretation</u>
EX 2	5/9/98	1	Scott/BCR, B	0/1, p/s, upper and mid. Peripheral upper lung infiltrates/fibrosis with probable peripheral granulomatous mass RUL. Calcified granulomata lung and mediastinal nodes. Change compatible with healed Tb $\pm$ activity. Em; hi; tb.
CX 4	5/11/98	1	Robinette/B	2/1, q/q, 6 zones; ax; large opacities, A; di; cn; em; id.
CX 1	5/11/98	1	DePonte/BCR, B	2/2, p/p, 6 zones; ax; large opacities, B; di.
CX 3	5/11/98	3	Fino/B	2/2, q/p, lower zones; ax.
EX 21	5/11/98	1	Wheeler/BCR, B	Ill defined oval 2 x 7 cm mass in lateral portion both upper lobes and lower a apices compatible with conglomerate Tb Tb with some small nodules and linear scars in upper lobes due to Tb unknown activity. Calcified granulomata in hilar nodes and few tiny ones in both mid and upper lungs from healed Tb. Histoplasmosis, giant cell arthritis and other inflammatory diseases could give this pattern but Tb most commonly involves upper lobes

and apices. A few small nodules could be pneumoconiosis but Tb best explains the asymmetry and location of masses. Low profusion of background nodules makes large opacities of CWP unlikely unless there is a well documented history of unprotected high dust exposure.



<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u>	<u>Interpretation</u>
EX 20	5/11/98	1	Scott/BCR, B	Focal infiltrates or ill-defined masses both apices compatible with Tb, unknown activity. Calcified granulomata both hilar regions and RM-U lung compatible with healed Tb.
EX 3	5/22/98	2	Wheeler/BCR, B	Unchanged except for technique since last exam 5/11/98.
EX 4	5/22/98	2	Scott/BCR, B	0/1, p/s, upper and mid. Calcified granulomata lungs and mediastinal nodes. Peripheral infiltrates/fibrosis upper lungs with probable granulomatous mass right apex. Changes compatible with healed Tb, cannot rule out activity. Em; hi; tb.
EX 5	10/21/98	1	Wheeler/BCR, B	Unchanged except for technique since last exam 5/12/98.
EX 6	10/21/98	1	Scott/BCR, B	0/1, p/s, upper and mid zones. Calcified granulomata lungs and mediastinum. Peripheral infiltrates/fibrosis and probable granulomatous masses upper lungs. Changes compatible with healed Tb, cannot rule out activity. Em; hi; tb.
EX 7	11/18/98	1	Wheeler/BCR, B	Unchanged since last exam 10/21/98.
EX 8	11/18/98	1	Scott/BCR, B	0/1, p/s, upper and mid zones. Calcified granulomata lungs and mediastinum. Peripheral infiltrates/fibrosis with probable peripheral granulomatous mass RUL. Changes compatible with healed Tb $\pm$ disease.

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u>	<u>Interpretation</u>
DX 13	12/1/98	1	Paranthaman/B	2/2, q/t, 6 zones; ax; large opacities, B; di; em (emphysema).
DX 14	12/1/98	2	Navani/BCR, B	2/1, q/p, 6 zones; ax; large opacities, B; di.
EX 17	12/1/98	2	Scott/BCR, B	Scattered calcified granulomata. Upper lung scarring/ infiltrates with hilar elevation. Probable granulomatous masses both apices. Changes compatible with Tb, partially healed. Cannot rule out active disease.
EX 18	12/1/98	2	Wheeler/BCR, B	Oval 4.5 cm mass in lower right apex and subapical portion RUL and 1.5 cm mass in lateral RUL and ill defined mass or fibrosis in lateral LUL between anterior ribs with ill defined infiltrate or fibrosis compatible with conglomerate Tb with small calcified granulomata in mid and upper lungs indicating at least some healed calcifications that may be in the masses which would indicate granulomatous disease more likely than tumors. Probable few areas of emphysema.  Silicosis and coal workers' pneumoconiosis are unlikely because disease is largely peripheral and apical and there are at least as many irregular scars as nodules.

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u>	<u>Interpretation</u>
EX 9	12/7/98	2	Wheeler/BCR, B	<p>Unchanged except for technique since last exam, 11/18/98. Mass-like fibrosis apices and posterior and lateral portion RUL &gt; LUL compatible with conglomerate Tb with linear scars extending to lateral pleura. Calcified granulomata in both mid and upper lungs and in left hilar adenopathy compatible with healed Tb. Em. Ih. Tb.</p> <p>Suggest CT scan. They are not large opacities of CWP because they are peripheral and lack the background q and r nodules from which large opacities merge. Also there is sparing of central portion mid and upper lungs where silicosis and CWP develop in symmetrical fashion. He is also young. The large opacities that I have seen are usually from WW2 drillers who worked without respiratory protection.</p>
EX 10	12/7/98	2	Scott/BCR, B	<p>0/1, p/s, upper and mid zones. Calcified granulomata lungs and mediastinum. Peripheral infiltrates/fibrosis upper with probable peripheral granulomatous mass RUL. Changes compatible with healed Tb, cannot rule out activity. Em; hi; tb.</p>

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/Qualifications</u>	<u>Interpretation</u>
EX 19	5/12/99	1	Wheeler/BCR, B	<p>Ill defined 2 x 5 cm mass in both lower apices and lateral subapical portion upper lobes with linear scars extending to pleural compatible with conglomerate Tb with calcified granulomata in hilar and right paratracheal nodes and small ones in both mid and upper lungs mixed with mainly inear and irregular scars or infiltrates compatible with Tb unknown activity, probably healed. Increased AP diameter chest partly due to kyphosis but check for emphysema.</p> <p>There could be a few nodules of pneumoconiosis largely hidden by other disease in upper lobes but Tb explains all the lung and pleural disease.</p>
DX 24	5/12/99	1	Scott/BCR, B	<p>Calcified hilar and mediastinal nodes and several calcified granulomata upper lungs. Fibrosis/infiltrates peripheral upper lungs. Possible granulomatous mass periphery RUL or dense infiltrate/fibrosis. Hilar elevation. Changes compatible with healed Tb, activity cannot be excluded.</p>
CX 6	3/16/00	1	Robinette/B	2/2, q/p, 6 zones; ax; large

opacities, B; di; cn; em.

<u>Ex.No.</u>	<u>Date of X-ray</u>	<u>Film Qual.</u>	<u>Physician/ Qualifications</u>	<u>Interpretation</u>
CX 6	3/16/00	-	Mullens	Nodular interstitial lung disease with upper lobe masses consistent with silicosis/CWP and progressive massive fibrosis.
EX 31	3/16/00	1	Scott/BCR, B	Peripheral infiltrates and/or masses apices. Hilar elevation. Calcified granulomata. Changes compatible with Tb, unknown activity. Hyperinflation lungs compatible with emphysema.
EX 30	3/16/00	1	Wheeler/BCR, B	Oval 6 x 3 cm mass RUL & 7 x 2.5 cm mass LUL with few linear scars, compatible with conglomerate Tb. Calcified granulomata, thin calcifications, few tiny calcified granulomata in both mid lungs and one in RLL, all compatible with healed Tb or histoplasmosis. Emphysema.  Egg shell calcifications in nodes were once considered to be from silicosis but healed granulomatous disease is far more common and may have been the cause in people who did have pneumoconiosis. In this case, there are no small symmet-

rical central nodules in upper lungs to suggest silicosis or CWP and the findings are typical of advanced untreated tuberculosis although other less common diseases can give the same appearance.

#### B. Biopsy Evidence

The claimant underwent a lung biopsy on May 12, 1998. Two pieces of tissue, placed on one slide, were examined. Dr. David R. Hudgens's microscopic description was:

One of the fragments contains a small nodule of hypocellular collagen surrounded by chronic inflammation and anthracitic histiocytes. The other biopsy consists of bronchial tissue with a few attached alveoli showing focal fibrosis and anthracosis. There is a single, poorly-preserved, multi nucleated cell which contains a nuclear inclusion which is eosinophilic and surrounded by a clear halo (possible Cowdry type A herpetic intra nuclear inclusion).

Dr. Hudgens's diagnoses were possible herpetic giant cell and fibrotic nodule. (CX 3).

Dr. P. Raphael Caffrey reviewed the one biopsy slide, Dr. Hudgens' report, and other medical reports on behalf of the employer. He issued a report on April 19, 1999. Dr. Caffrey's review of the slide revealed:

a moderate amount of anthracitic pigment with one micronodule. No macules were identified. The one micronodule would be consistent with a micronodule simple coal worker's pneumoconiosis.

I cannot make a diagnosis of complicated pneumoconiosis because the biopsy material definitely does not have findings consistent with complicated pneumoconiosis. I cannot properly or objectively interpret the x-ray findings because even though there are described large opacities in the lungs, one cannot be sure what these large opacities represent. Mr. Stanley, for example, has a significant smoking history of one pack a day for 39 years and according to Dr. Paranthaman's medical report of 12-1-98 he was still smoking.

From these reports, the blood gas studies, the x-rays, the pulmonary function studies, etc., Mr. Stanley does have apparently enough respiratory impairment that would keep him from performing manual labor in a coal mine. How much of this

respiratory disability is due to the coal mine dust he may well have inhaled and how much is due to his years of smoking cigarettes, I cannot say objectively.

(DX 23). Dr. Caffrey is board-certified in anatomical and clinical pathology. (EX 29).

C. Pulmonary Function Studies

<u>Date</u>	<u>Ex. No.</u>	<u>Age/Hgt.</u>	<u>FEV1</u>	<u>FVC</u>	<u>FEV1/ FVC</u>	<u>MVV</u>	<u>Coop/ Comp.</u>
1/12/83	EX 22	33/66"	3.61 *3.73	5.14 4.94	70% 75%	102 --	Good/ Good
7/13/95	DX 23	46/66"	2.59 *2.77	4.48 4.64	58% 60%	72 73	--
4/28/98	DX 21, CX 3	49/66"	2.46 *2.18	4.30 4.00	57% 55%	68 62	Good/ Good
5/22/98	DX 21, CX 3	49/66"	3.02	4.66	65%	116	Good/ Good
12/1/98	DX 11	49/65"	2.54	4.65	54.54%	98	Good/ Good
12/7/98	DX 21	49/66"	2.55	4.39	58%	96	Good/ Good
5/12/99	DX 24	50/67"	3.00 *3.09	5.05 4.99	59% 62%	98 --	Good/ Good
3/16/00	CX 6	51/66"	2.77	4.54	61%	--	Good/ Good

\* Results obtained post-bronchodilator.

D. Arterial Blood Gas Tests

<u>Date</u>	<u>Physician</u>	<u>pCO2</u>	<u>pO2</u>	<u>Ex. No.</u>
-------------	------------------	-------------	------------	----------------

1/11/83	Smiddy	36.1	77.7	EX 26
7/13/95	Hall	38.1	65.6	DX 23
12/1/98	Paranthaman	38	83	DX 12
5/12/99	McSharry	37	83	DX 24
3/16/00	Robinette	39.7	79.0	CX 6

E. CT scans and Medical Opinions

The records of St. Mary's Hospital were submitted into the record. Pertinently, they indicate that the claimant was hospitalized in May 1998 for pneumonia. Tuberculosis was suspected; the sputum tests were negative. The x-ray readings and pulmonary function test results from St. Mary's are listed above. Dr. DePonte's review of the fluoroscopy indicated that the biopsy was obtained from the right lung apex, in the region of the conglomerate mass. Diagnoses in the hospital records include chronic obstructive pulmonary disease (COPD) with pulmonary fibrosis/ CWP. (CX 3).

Dr. Joseph F. Smiddy examined the claimant on May 13, 1998 on referral from Dr. Thomas Cortellesi. Dr. Smiddy reviewed the claimant's histories, symptoms, and medications. The claimant was not smoking at that time, wearing a Nicoderm Patch. The family history was positive for tuberculosis in an uncle.<sup>2</sup> Examination revealed scattered rales and rhonchi, as well as a prolonged expiratory phase. Dr. Smiddy noted that "[t]he patient carried in hand chest x-rays showing advanced complicated coal workers pneumoconiosis. The films are copies and I cannot rule-out an additional concomitant process." A follow-up appointment was scheduled for testing. (CX 2). Dr. Smiddy is board-certified in internal medicine and board-eligible in pulmonary medicine. (EX 29).

The claimant returned to Dr. Smiddy's office on May 22, 1998. A pulmonary function study revealed a moderate obstructive ventilatory defect with normal DLCO. The claimant's chest x-ray continued to show changes of complicated pneumoconiosis. The biopsy results were also reviewed. Dr. Smiddy continued the claimant's medications and again advised the claimant to stop smoking. He referred the claimant back to Dr. Cortellesi for continuing care. (CX 2).

---

<sup>2</sup> This history is not inconsistent with the claimant's statement to Dr. Robinette that he had no known exposure to tuberculosis. The claimant did not state that he had contact with his uncle.



Dr. S.K. Paranthaman examined the claimant on December 1, 1998. He reviewed the claimant's histories, symptoms, and medications. The claimant had pneumonia two times in 1998, and was also hospitalized for a case of pneumonia ten years earlier. He experienced attacks of wheezing during the two previous years. The claimant was currently smoking one pack of filtered cigarettes per day. Examination revealed decreased breath sounds bilaterally. An x-ray was positive for complicated pneumoconiosis, 2/2, q/t, ax. A resting EKG was within normal limits, as was a resting arterial blood gas study. A pulmonary function study was also obtained. Dr. Paranthaman diagnosed complicated coal workers' pneumoconiosis related to the claimant's coal mine employment. He stated that "[t]here is mild airway obstruction in the spirogram. Arterial blood gas at rest is normal indicating that the functional impairment related to coal workers' pneumoconiosis is mild. However, because of the presence of large opacities in both upper lobes of the category B, he is considered to be totally disabled to do the coal mine occupation such as drill operator or hauler operator." (DX 11).

Dr. Roger J. McSharry is board-certified in internal, pulmonary, and critical care medicine. He examined the claimant on May 12, 1999 on behalf of the employer. Dr. McSharry reviewed the claimant's histories, symptoms, and medications. His examination revealed clear lungs. An electrocardiogram was normal, as was a pulmonary function study. A resting arterial blood gas test was normal except for an elevated carboxyhemoglobin level. An x-ray was interpreted by Dr. Scott as showing bilateral upper lobe masses and increased markings consistent with volume loss; a number of small opacifications, calcified mediastinal and hilar lymph nodes; and some hyperinflation of the lower lung zones without opacifications. A CT scan was interpreted by Dr. McSharry as showing bilateral upper lobe irregular masses with associated increased nodular markings, traction on the surrounding lung parenchyma, and some evidence of emphysema in the lung bases. Calcification of lymph nodes in the hilum and mediastinum was confirmed. Dr. Caffrey's pathology report was reviewed. Dr. McSharry concluded that:

Mr. Stanley certainly has significant abnormalities in his lungs. It is exceedingly unlikely these are due to coal mining. My justification for this statement is as follows:

The pulmonary function tests document mild obstructive lung disease and his clinical history of chronic bronchitis confirms this. The fact that bronchodilators have been beneficial indicates asthma plays a role in his respiratory symptoms. Exposure to coal dust does not cause this pattern of pulmonary abnormality, while smoking is a common cause of obstructive lung disease.

He does have breathlessness symptoms, but the degree of lung abnormality demonstrated by PFT is quite mild. I would expect this to result in symptoms only with heavy physical labor. The pattern of abnormality seen on the pulmonary function test is not what would be expected in coal worker's pneumoconiosis or silicosis, as there is no evidence of restrictive lung disease. The resting arterial blood gases are normal and do not suggest significant lung pathology.

The abnormality on the chest x-ray and CT scan of the chest can be explained either by infectious granulomatous disease (such as histoplasmosis or tuberculosis) or less convincingly by pneumoconiosis. It would be distinctly unusual to have conglomerate masses of pneumoconiosis without a background of opacities seen in pneumoconiosis. These opacities were specifically excluded by Dr. Scott's interpretation of the x-ray.

The biopsy specimen cannot answer the question of whether the upper lobe densities are masses of complicated pneumoconiosis, as the masses were not biopsied. The single micro-nodule noted in the specimen does not in and of itself allow a diagnosis of coal worker's pneumoconiosis to be made, although it is compatible with that diagnosis.

Based on the evidence above, I conclude that Mr. Stanley has the respiratory capacity to perform his last job as a drill operator as it was described to me. The abnormalities seen on pulmonary function tests do not in my opinion bear any relation to his history of working around coal. The radiographic abnormalities demonstrated cannot be exclusively attributed to any particular cause, but seem most likely a result of respiratory infection and would be distinctly unusual to result from coal worker's pneumoconiosis.

(DX 24).

Dr. Paul S. Wheeler reviewed the CT scan obtained by Dr. McSharry on May 12, 1999. His opinion was advanced tuberculosis, probably healed, in upper lobes, apices, and superior segments lower lobes with conglomerate masses and extensive calcified granulomata in mediastinal and hilar nodes. He also found emphysema including areas of centrilobular emphysema. The masses measured 4 and 3 centimeters. Consistent with his x-ray readings, Dr. Wheeler stated that:

There could be a few small nodules of pneumoconiosis but the predominant small opacity is irregular and linear. The pattern is asymmetrical with peripheral and apical disease compared to silicosis and CWP which gives symmetrical nodules in central portion mid and upper lungs. Histoplasmosis can cause extensive hilar and mediastinal calcified granulomata but Tb is much more likely to attack upper lobes and apices.

Historically, most large opacities of pneumoconiosis have been in drillers during and prior to WW2 working with no protection. This man was born after that era.

Tb that is self-cured or inadequately treated leaves scars, masses and calcified granulomata. Properly treated, even advanced active Tb can clear completely with proper therapy.

(EX 28).

Dr. James R. Castle is board-certified in internal and pulmonary medicine. (EX 29). He reviewed the claimant's medical records and issued a report on November 22, 1999. From those records, Dr. Castle concluded that the claimant "may have pathologic evidence of minimal simple coal workers' pneumoconiosis." He explained that:

At no time did [the claimant] demonstrate any physical findings indicating the physical presence of an interstitial pulmonary process such as would be expected with coal workers' pneumoconiosis. He did not ever demonstrate the finding of rales, crackles, or crepitations. His pulmonary examination has been essentially unremarkable.

There has been a very marked degree of variation in interpretations of his chest x-rays. Several radiologists felt there was evidence of simple and complicated coal workers' pneumoconiosis present radiographically. Numerous other radiologists including those from John Hopkins University felt there was no evidence of pneumoconiosis and especially no evidence of complicated pneumoconiosis. These opinions were further buttressed by the CT scan findings which indicated changes most likely related to old granulomatous disease. Drs. Scott, Wheeler and McSharry all explained in detail why the radiographic changes were most likely due to granulomatous disease rather than coal workers' pneumoconiosis. He also had evidence of calcified granulomata present on both the x-rays and a CT scan.

The physiologic studies that have been done have shown either a mild or moderate degree of airway obstruction without restriction or diffusing capacity abnormality. The degree of obstruction has fluctuated between mild to moderate over a period of time. There has been a marked degree of variability in the FVC and FEV1 over time. This is inconsistent with a diagnosis of coal workers' pneumoconiosis. This is especially inconsistent with a diagnosis of complicated coal workers' pneumoconiosis. Certainly if one had evidence of category B pneumoconiosis, the physiologic changes would be those of very significant, *irreversible* obstructive and *restrictive* lung disease. If the abnormalities present on chest x-ray were indeed due to pneumoconiosis these findings would certainly be expected physiologically. That was not the case. What was found was markedly variable mild-moderate airway obstruction without restriction or diffusion abnormality. These findings are not consistent with complicated coal workers'

pneumoconiosis. These findings are consistent with and indicative of tobacco smoke induced chronic airway obstruction with a significant asthmatic component to this.

The arterial blood gases that were done were normal. There was no evidence of blood gas transfer abnormalities. This is not the finding one would expect with complicated coal workers' pneumoconiosis.

A single, small lung biopsy showed evidence of one single micronodule that Dr. Caffrey indicated was consistent with coal workers' pneumoconiosis. Although this single finding may indicate the presence of simple coal workers' pneumoconiosis pathologically, it does not indicate that the other findings present are due to coal workers' pneumoconiosis or complicated coal workers' pneumoconiosis. None of the other findings in this case corroborate or substantiate this finding. The biopsy did not confirm the presence of complicated coal workers' pneumoconiosis.

Dr. Castle also concluded that the claimant has the respiratory capacity to resume his former coal mine employment. He attributed the mild to moderate degree of respiratory impairment to asthmatic bronchitis and chronic obstructive pulmonary disease caused by smoking. (EX 27).

Dr. Emory Robinette is also board-certified in internal and pulmonary medicine. (CX 5). He examined the claimant on March 16, 2000, and reviewed his histories, symptoms, and medications. At the time, the claimant was taking a Z-PAK for an acute bronchitic exacerbation of his lung disease and he was smoking 3 to 4 cigarettes per day. The claimant's father and three brothers died of cancer. The claimant denied any exposure to tuberculosis. Examination revealed diminished breath sounds with poor air movement, and bilateral inspiratory crackles. An x-ray showed diffuse interstitial fibrosis with conglomerate masses in the upper lungs. There were areas of progressive massive fibrosis with calcifications in the left perihilar regions, distortion of the hilar regions and emphysematous type change. Dr. Robinette classified the findings as 2/2, q/p, B, axillary coalescence, calcification, distortion, and emphysema. A pulmonary function study was essentially normal. Lung volume studies were normal. The diffusion capacity was impaired at 58% of predicted. A resting arterial blood gas test was normal. The carboxyhemoglobin level was 5.5%. A resting EKG showed a normal sinus rhythm with a nonspecific ST-T wave change present. Poor R wave progression was noted in leads V2 and V3 which were nonspecific. A comprehensive metabolic profile was also normal.

Dr. Robinette diagnosed complicated coal workers' pneumoconiosis with progressive massive fibrosis; chronic bronchitis, probably industrial in nature; degenerative arthritis; and hypothyroidism on replacement therapy. He stated that:

The chest x-ray showed radiographic evidence of diffuse nodular interstitial lung disease consistent with an underlying diagnosis of an occupational pneumoconiosis. There were diffuse fine opacities throughout all lung zones with evidence of axillary coalescence and

areas of progressive massive fibrosis. There was no history of TB exposure in the past and he denies any constitutional symptoms consistent with Mycobacterial disease such as weight loss, fever, sweats, or malaise.

It is recognized that there is a significant concern about concurrent silica exposure in miners who work as roof bolter, shaft development workers or drill operators. These individuals are more predisposed to areas of progressive massive fibrosis.

Although Mr. Stanley has a normal FEV1 and FVC there is a significant reduction of his diffusion capacity. The medical literature documents that gas exchange abnormalities occur in miners with coal workers' pneumoconiosis and that in general individuals who have diffuse small opacities scattered throughout the lungs may be susceptible to an impairment of the diffusion capacity. Obviously, in a patient with progressive massive fibrosis they may have further reduction of the diffusion capacity as a result of destruction in the alveolar capillary surface areas.

It is my medical opinion that Mr. Stanley is disabled from working on the basis of his occupational pneumoconiosis and this condition is probably progressive nature and maybe associated with progressive decline of his lung function. This condition is directed (sic) related to his prior coal mining employment and unrelated to any smoking history.

(CX 6).

After reviewing additional medical records, namely Dr. Robinette's reports and two B/BCR readings, Dr. McSharry issued a supplemental report on April 26, 2000. He stated that his opinion remained the same, and commented that:

Although there are some differences in the objective pulmonary function testing compared with the studies performed in my office last year, I find that in general the same pattern of abnormality is seen, namely normal spirometric values and lung volumes as well as fairly normal arterial blood gas. The diffusion capacity measured by Dr. Robinette's office is somewhat lower, but this abnormality is consistent with the scarring seen on his x-rays.

(EX 32).

Also after reviewing the additional medical records, Dr. Castle issued a supplemental report on May 8, 2000. His opinion remained the same that the Claimant does not have complicated coal workers' pneumoconiosis although, based on the biopsy, he may have simple pneumoconiosis. He also

commented that "[t]here was evidence of very mild airway obstruction and a minimal decrease in the diffusing capacity after correction for alveolar volume. These changes are not those that are typical of [simple and complicated] coal workers' pneumoconiosis." (EX 33).

## DISCUSSION AND APPLICABLE LAW

Because the claimant filed his application for benefits after March 31, 1980, this claim shall be adjudicated under the regulations at 20 C.F.R. Part 718. Under this part of the regulations, the claimant must establish by a preponderance of the evidence that he has pneumoconiosis, that his pneumoconiosis arose from coal mine employment, that he is totally disabled, and that his total disability is due to pneumoconiosis. Failure to establish any of these elements precludes entitlement to benefits. *See Anderson v. Valley Camp of Utah, Inc.*, 12 BLR 1-111, 1-112 (1989).

### Pneumoconiosis and Causation

Under the Act, pneumoconiosis is defined as a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. 30 U.S.C. § 902(b). Section 718.202(a) provides four methods for determining the existence of pneumoconiosis: X-ray evidence, biopsy or autopsy evidence, application of a presumption, and medical opinion evidence. §§ 718.202(a)(1)-(4).

As to biopsy evidence, § 718.106(c) specifically provides that "[a] negative biopsy is not conclusive evidence that the miner does not have pneumoconiosis. However, where positive findings are obtained on biopsy, the results will constitute evidence of the presence of pneumoconiosis." 20 C.F.R. § 718.106(c).

I find that the claimant has established that he has simple pneumoconiosis. While highly qualified readers disagreed as to whether the changes seen on the x-rays and CT scans were consistent with pneumoconiosis or a granulomatous disease such as tuberculosis, the biopsy revealed a micronodule which was consistent with simple coal workers' pneumoconiosis. Drs. Hudgens and Caffrey, the only pathologists to review the tissue, agreed that the micronodule was present. Neither pathologist attributed the micronodule to a cause other than coal dust. Both remarked on the presence of anthracosis or anthracitic pigment in the tissue. Therefore, even though the tissue biopsied was small, it does establish that the claimant has coal workers' pneumoconiosis.<sup>3</sup> §718.202(a)(2).

Additionally, I find that the weight of the x-ray evidence, even standing alone, is positive for changes consistent with pneumoconiosis. Drs. Wheeler and Scott concluded that the changes were

---

<sup>3</sup> In keeping with the spirit of § 718.106(c), however, the biopsy is not conclusive evidence that the claimant does not have tuberculosis as well, a point also made by Dr. Caffrey.

most likely representative of tuberculosis and listed factors supporting their conclusion. However, in reviewing one x-ray, Dr. Wheeler commented that "there are at least as many irregular scars as nodules," indicating that the x-rays showed a significant amount of rounded nodules, the latter being the type of nodule which is attributable to classical/medical pneumoconiosis. Further, the preponderance of the x-ray evidence indicates that both irregular and rounded opacities can be classified as consistent with pneumoconiosis, as seen in the readings of equally qualified physicians. Dr. Scott classified some of the opacities as 0/1. He and Dr. Wheeler apparently chose to not classify any of the opacities as consistent with category 1 or higher pneumoconiosis because they focused on the issue of causation. Yet it must be remembered that the statutory definition of pneumoconiosis at §718.201 includes silicotuberculosis, so that physicians cannot limit themselves to the characteristics associated with classical/medical pneumoconiosis when interpreting an x-ray under the Act. Significantly, no reader or physician stated that tuberculosis could not be or was not related to coal mine employment, although Dr. Wheeler acknowledged that pneumoconiosis (silicosis) and granulomatous disease have been seen together and that egg shell calcifications have been attributed to both disease processes. §§ 718.202(a)(1), 718.203(b).

Moreover, no other medical evidence supports a diagnosis of tuberculosis. The sputum test was negative, as was the biopsy. The hospital treated the claimant for pneumonia. Dr. Robinette discussed the possible symptoms of tuberculosis with the claimant, and an episode of tuberculosis was implicitly ruled out. Dr. Smiddy, who assisted in the treatment of the claimant, also did not diagnose tuberculosis. The hospital, Dr. Robinette, and Dr. Smiddy all came to the conclusion of coal workers' pneumoconiosis. That the claimant was exposed to coal dust is undisputed.

While the employer's experts also speculated that the claimant could have a different granulomatous disease process other than tuberculosis, again there is no other medical evidence to support such a diagnosis.

The findings on the physical examinations by Drs. Smiddy, Paranthaman, McSharry, and Robinette were essentially the same, with the physicians's different conclusions resting on the interpretations of the x-rays, a debate Dr. Castle also joined in. Drs. Smiddy, Paranthaman, and Robinette diagnosed coal workers' pneumoconiosis. Drs. McSharry and Castle did not. Nevertheless, the latter two physicians acknowledged that the biopsy was consistent with coal workers' pneumoconiosis. None of the physicians who diagnosed coal workers' pneumoconiosis factored in the mild obstruction, thus rendering Drs. McSharry's and Castle's points about the variance in FEV1 and FVC, the response to bronchodilators, and the effects of smoking, essentially immaterial. And contrary to Dr. Castle's summation that the claimant never demonstrated rales, Dr. Smiddy heard rales on examination. Considering these factors, and that I have found the biopsy and x-ray evidence to be positive for pneumoconiosis, I give greater weight to the opinions of Drs. Smiddy, Paranthaman, and Robinette. As such, I find pneumoconiosis established by the preponderance of the medical opinion evidence. § 718.202(a)(4).

Finally, under § 718.202(a)(3), a claimant may also prove the existence of pneumoconiosis if one of the presumptions at §§718.304 to 718.306 applies. The presumptions at §§ 718.305 and 718.306 are inapplicable because they only apply to claims that were filed before January 1, 1982, and June 30, 1982, respectively. Section 718.304 provides an irrebut-able presumption of total disability due to pneumoconiosis based on x-ray, biopsy, or equivalent evidence of complicated pneumoconiosis.

The x-rays undisputedly show large masses, which are classifiable as large opacities "B", although as with the issue of simple pneumoconiosis, the readers differed as to whether the opacities are consistent with complicated pneumoconiosis. The biopsy itself was not of one of the masses, and therefore cannot provide a diagnosis of complicated pneumoconiosis. However, I do note that the biopsy was obtained from tissue located by one of the masses, and therefore does reflect on the tissue in the background of the mass. That background included a micronodule and anthracitic pigment/anthracosis. The medical opinions are divided on the issue of complicated pneumoconiosis as they are on simple pneumoconiosis.

Weighing all of the evidence on complicated pneumoconiosis, I find that the disease has been established. The claimant has a background of simple coal workers' pneumoconiosis from which the complicated pneumoconiosis would have formed. There are irregular and rounded opacities, including of the "q" type. The x-rays show that the advanced changes were present as early as 1995, before the claimant ceased his coal mine employment. One of the claimant's positions was that of a drill operator, and his credible testimony shows that he was unable to wear a respirator most of the time and was heavily exposed to dust. For these reasons, and the reasons given for my earlier finding of simple pneumoconiosis versus a granulomatous process, I find that the claimant has established complicated pneumoconiosis. He is therefore entitled to invocation of the § 718.304 irrebutable presumption of total disability due to pneumoconiosis.

Based on the above, I find that the claimant has established that he has simple pneumoconiosis. §§ 718.202(a)(1), (2), (4). I also find that his pneumoconiosis arose from his coal mine employment. § 718.203(b), Employer's Closing Argument. I additionally find that the claimant has complicated pneumoconiosis and is disabled therefrom. §§ 718.304, 718.202(a)(3).

#### Total Disability and Causation

While invocation of the § 718.304 presumption of total disability due to pneumoconiosis entitles the claimant to benefits, I will nevertheless address the other evidence related to total disability for the benefit of the parties.

None of the pulmonary function studies produced qualifying values. Therefore, the claimant has not established total disability under § 718.204(c)(1). As none of the arterial blood gas tests produced



qualifying values, the claimant has not established total disability under § 718.204(c)(2). The record does not contain any evidence of cor pulmonale with right-sided congestive heart failure. Therefore, the claimant cannot establish total disability under § 718.204(c)(3).

No physician opined that the claimant was totally disabled from a pulmonary or respiratory standpoint based on any of the pulmonary function or arterial blood gas tests, or their examination findings. The opinions of total disability are based on the finding of complicated pneumoconiosis, with the presumption that the claimant is totally disabled, or with the belief that the complicated pneumoconiosis will ultimately render him totally disabled. Thus, the medical opinions do not establish total disability apart from the § 718.304 presumption. § 718.204(c)(4).

Based on the foregoing, I find that the claimant has not established total disability apart from invocation of the §718.304 presumption of total disability. However, again, that presumption is irrebutable.

#### Attorney's Fee

Claimant's counsel has thirty days to submit an application for an attorney's fee. The application shall be prepared in strict accordance with 20 C.F.R. §§ 725.365 and 725.366. The application must be served on all parties, including the claimant, and proof of service must be filed with the application. The parties are allowed thirty days following service of the application to file objections to the fee application.

#### ORDER

Humphrey's Enterprises, Inc., and its carrier, CNA Insurance Company, is hereby ORDERED to pay the following:

(1) To the claimant, Alvin H. Stanley, all benefits to which he is entitled under the Act, augmented by reason of his three dependents, commencing November 1, 1998, the month in which the claim for benefits was filed;

(2) To the claimant, all medical and hospitalization benefits to which he is entitled, commencing November 1, 1998;

(3) To the Secretary of Labor, reimbursement for payments the Secretary has made to the claimant under the Act. The employer may deduct such amounts, as appropriate, from the amounts the employer is ordered to pay under paragraphs 1 and 2 above; and

(4) To the Secretary of Labor or to the claimant, as appropriate, interest computed in accordance with the provisions of the Act or regulations.

---

JOSEPH E. KANE  
Administrative Law Judge

NOTICE OF APPEAL RIGHTS: Pursuant to 20 C.F.R. §7 §725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this Decision and Order by filing a Notice of Appeal with the Benefits Review Board at P.O. Box 37601, Washington, D.C. 20013-7601. A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Room N-2117, 200 Constitution Avenue, N.W., Washington, D.C. 20210.